CASE REPORT

Near fatal iron intoxication managed conservatively

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SUMMARY

We report here the case of an infant who presented to the emergency department after unintentional ingestion of almost 130 mg/kg of elemental iron. The infant had evidence of serious toxicity in the form of hypotension, metabolic acidosis and excessive irritability. Her serum iron levels were 360 µg/dl, which was well above the normal range for her age. Despite such high serum levels, the infant made an uneventful recovery with medical management alone and did not require exchange transfusion. We chose to report this case to highlight her uneventful recovery with conservative therapy alone.

BACKGROUND

Acute unintentional iron poisoning is a common and potentially fatal intoxication in children below 5 years of age. A report from the American Association of Poison Control Centres revealed that iron supplements were the single most frequent cause of paediatric unintentional ingestion fatalities, accounting for 30.2% of reported paediatric pharmaceutical unintentional ingestion fatalities over an 8-year period.¹ Iron preparations are very commonly administered to pregnant and lactating mothers, and thus toddlers in such households are easy victims of poisoning.

Although iron is a therapeutic drug in recommended dosages, excessive iron in the free state is capable of producing toxicity by affecting multiple cellular processes by catalysing redox reactions with lipid peroxidation and free radical formation.² The severity of intoxication depends on the amount of elemental iron ingested. Serious toxicity is usually associated with a dose of >40 mg/kg of elemental iron. Levels more than 100 mg/kg are almost always fatal.³ Only a few cases of survival with such high levels have been reported, and most of these cases required exchange transfusion.^{4–6}

We report here the case of an infant who survived after ingestion of 130 mg/kg of elemental iron. We chose to report the case to highlight the remarkable survival of this infant with supportive care alone.

CASE PRESENTATION

A 10-month-old female child weighing 8 kg presented to the emergency department (ED) with an alleged history of consumption of 13 iron tabs (of 200 mg strength with 60 mg elemental iron each, total dose ingested=780 mg or 130 mg/kg), which was prescribed to her mother for her pregnancy. The child presented to the ED after 3.5 h with complaints of recurrent vomiting and increasing irritability. The vomiting continued in the ED, and

the vomitus contained a red coloured unabsorbed drug. She was irritable with signs of severe dehydration in the form of decreased skin turgor, sunken eyes and dry oral mucosa. Her heart rate was 110/min, respiratory rate was 44/min without any evidence of increased work of breathing, capillary refill time was 2–3 s and her blood pressure was just below the fifth centile for her age (68/40 mmHg). The rest of the systemic examination was unremarkable.

INVESTIGATIONS

Her initial investigations revealed normal blood glucose levels (random blood sugar –120 mg/dl) with metabolic acidosis with respiratory compensation. The pH was 7.51, PCO₂ 15.4 mm Hg, PaO₂ 128 mm Hg, HCO₃⁻ 12.5 mmol/l and the base deficit 12.1. Her serum electrolytes and renal function tests were normal. The x-ray of her abdomen was normal. The serum iron levels obtained were 360 µg/dl. After 12 h of starting deferoxamine, the iron levels fell to 90 µg/dl, which was in the normal range (50–150 µg/dl). The metabolic acidosis also showed improvement as the repeat blood gas revealed a pH of 7.45, PCO₂ of 23.4, HCO₃⁻ of 17 and base deficit of –6.

TREATMENT

The child was immediately administered oxygen with a non-rebreathing face mask and started on fluid therapy for severe dehydration. She was also given intravenous antiemetics and antacids for her gastric irritation. Subsequently, she was shifted to the paediatric intensive care unit (ICU) within 30 min of her arrival where she underwent whole bowel irrigation with polyethylene glycol at a dose of 30 ml/kg/h till such time that she had started passing clear stools. This took about 3 h duration. She was also started on definitive therapy with intravenous deferoxamine within about 2 h of her admission at a dose of 15 mg/kg/h.

Although the dose of iron ingested was lethal² and the child had evidence of serious toxicity in the form of vomiting, severe dehydration with metabolic acidosis, we decided to give her a trial of conservative therapy with deferoxamine and supportive care as she was alert and interactive and her serum iron levels were only in the serious toxicity range (350–500 µg/dl) and not in the fatal range (ie, >1000 µg/dl).² We had also planned for exchange transfusion and made arrangements for the same in case of any worsening clinically or biochemically. However, after 6 h of therapy, the child had become normovolaemic and started accepting breast feeds. Therefore, the same management was continued. Deferoxamine was administered for a

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total duration of 12 h. After this repeat, iron levels were obtained which were in the normal range, and therefore deferoxamine was discontinued. The child showed no adverse reactions to iron chelation therapy during her stay.

OUTCOME AND FOLLOW-UP

The child was monitored for the next 48 h for any complications that might arise and was discharged after 72 h of ICU stay after she made complete recovery.

DISCUSSION

Iron is toxic in the free state and therefore, in the body, it binds to proteins such as ferritin and transferrin to avoid tissue damage.³ In situations of overdose, the binding capacity of these proteins is overwhelmed, and thus iron becomes free to exert its action on various organs and organ systems with hazardous consequences. It commonly affects the gastrointestinal mucosa causing erosions, the vascular system causing vasodilatation, increased capillary permeability, metabolic acidosis and cell injury in various organs causing cell death.^{2 3} Clinically, iron poisoning may manifest as vomiting, diarrhoea, abdominal pain and, occasionally, significant blood loss.

A dose >30 mg/kg of elemental iron can be potentially toxic.² The case presented here had ingested an almost lethal dose of >100 mg/kg and presented with evidence of serious toxicity such as metabolic acidosis and shock.^{2 3} Normally, with ingestion of dosages of >100 mg/kg, the serum-free iron levels expected would be in the thousands. However, despite ingesting such high doses of the drug, the iron levels were not as high as expected. Persistent vomiting after ingestion could have played a major role in her serum iron levels remaining below those requiring exchange transfusions as most of the drug would have been eliminated in the process.

Iron poisoning has four clinical stages. Features of acute gastrointestinal irritation dominate the period up to 6 h after ingestion, and most patients do not develop other features or progress beyond this stage. Hypotension may arise at this stage either due to hypovolaemia or due to blood loss.^{2 3 7} In the index case, it was probably due to hypovolaemia. Severe poisoning is characterised by impairment of the sensorium, convulsions and metabolic acidosis. The child reported here was probably in stage 1 with evidence of serious toxicity in the form of increasing irritability and metabolic acidosis. In the second stage, which starts about 6–12 h after ingestion, the clinical features start showing remission. Few patients may progress to stage 3, which is 12–48 h after ingestion, wherein there is recurrence of symptoms in the form of shock and metabolic acidosis. Acute kidney injury and hepatocellular failure may develop in a few patients during this stage. The last (fourth) phase, which usually develops in young children 2–6 weeks after ingestion, is characterised by the recurrence of vomiting due to gastric or duodenal stenosis caused by the healing of gastric erosions.^{2 3 7}

Acute iron poisoning in humans has not been adequately studied because of the infrequent and sporadic occurrence of cases. Evidence-based management is therefore lacking and the management is thus case based. Assessment of severity of poisoning is essential for optimum management, but is difficult as the amount of elemental iron ingested cannot be precisely estimated in young children because of vomiting and diarrhoea. The commonly encountered clinical features may also be unreliable. However, the presence of shock, coma and metabolic

acidosis may indicate severe poisoning, and they have been shown to be poor prognostic factors for survival.⁷

The definitive therapy for iron poisoning is deferoxamine, an iron chelator that has been used over the past five decades as a therapeutic agent for the treatment of both acute and chronic iron overdoses.⁷ The recommended maximal dosage of deferoxamine is 80 mg/kg over 24 h. However, 100 mg of deferoxamine chelates only 8.5 mg of iron. This dose is often insufficient in severe iron poisoning. Doses as high as 210 mg/kg have been used in the past without any adverse reactions.⁶ Therefore, in this child also, we administered deferoxamine at a dose of almost 180 mg/kg/day, which is way above the recommended dose. Fortunately, there were no side effects of deferoxamine such as pain abdomen, nausea, diarrhoea, etc and no evidence of renal or hepatic disturbances up to 48 h after therapy.⁸ Apart from this, supportive care is the mainstay of iron poisoning. Few cases of severe iron intoxication have been managed with exchange transfusion in the past, and this remains a therapeutic option in children with severe toxicity.^{4–6}

Learning points

- ▶ Acute iron poisoning is a life-threatening emergency in infants.
- ▶ In infants and young children, the serum iron levels may not always correlate with the dosage ingested due to vomiting or diarrhoea in some cases.
- ▶ Management comprises of supportive care and deferoxamine therapy with dosages sometimes exceeding the recommended dosage of 80 mg/kg in cases of serious intoxication.
- ▶ Treatment of iron poisoning should be guided by serum iron levels rather than the dose ingested in young children.
- ▶ Parents should be educated time and again about the importance of prevention of such injuries as these are best prevented.

Competing interests None.

Patient consent Obtained.

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